Environmental Exposure to Tobacco Smoke and Lung Function in Young Adults[1-3]

MARIA ARDITO MASI,[4] JAMES A. HANLEY, PIERRE ERNST, and MARGARET R. BECKLAKE[5]

SUMMARY The relationship between lung function and environmental exposure to tobacco smoke (passive smoking) was studied in 293 nonsmoking young men and women, 15 to 35 yr of age. A self-administered mailed questionnaire was used to assess the lifetime environmental exposure to cigarette smoke at home and at work for each subject. Lung function information used here had been gathered in the course of a previous study of the determinants of lung function in early adulthood. In men, maximal midexpiratory flow rate (FEF_{25-75}) decreased in relation to an index of cumulative lifetime environmental exposure to tobacco smoke at home, after taking into account the effects of cumulative exposure at work as well as age, height, body size, respiratory pressures, and cooking fuels used at home. The components of this exposure index most closely related to the reduction in FEF_{25-75} were maternal smoking habits and exposure to second-hand smoke during childhood. In women, the diffusing capacity of the lung (D_{LCO}) decreased in relation to cumulative exposure to tobacco smoke at work, after accounting for the effects of cumulative lifetime exposure at home and the other factors mentioned above. These findings contribute to the gathering evidence that environmental exposure to tobacco smoke is harmful to respiratory health, and suggest that the effects are not insignificant. For instance, the FEF_{25-75} of a young man 20 yr of age who had never smoked and always lived at home would be 800 ml less if both his parents smoked than if they did not. Similarly, a young woman who had never smoked but had worked in an office for 10 yr where smoke was always seen or smelled would have a D_{LCO} 3 units lower than if she had worked in a smoke-free office.

AM REV RESPIR DIS 1988; 138:296-299

Introduction

The quality of indoor air (1) has emerged as one of the principal health concerns of the drive to energy conservation (2), particularly in northern latitudes where a harsh winter climate leads to strenuous efforts to diminish the dissipation of heat. A common and important source of indoor pollution is tobacco smoke, in particular, cigarette smoke (2), and Canada is among the world leaders in per capita cigarette consumption (3). Adverse effects of environmental exposure to tobacco smoke, also called passive smoking, have been demonstrated in most (4-12) but not all (13-15) studies of lung function in children. In adults, effects have been inconsistently found, but they may be more pronounced in subjects older than 40 yr of age (16-20). Adverse effects appear to be less readily demonstrable in warm dry climates (14, 15, 21). On balance, the evidence suggests dose-response relationships between exposure and any adverse effect on lung function, although estimates of exposure have been far from quantitative (22, 23). The objective of the present study was to determine whether cumulative lifetime environmental exposure to tobacco smoke in the home and/or at work affects the lung function of young adults 15 to 35 yr of age. This age group has not previously been the target of investigation, and such cross-sectional data as exist do not point to adverse lung function effects (16, 18). This is somewhat surprising since a recent longitudinal study suggests that passive exposure to maternal cigarette smoke reduced the rate of lung function growth of young persons 4 to 28 yr of age (7). In the present research, an effort was made to develop a cumulative index of lifetime exposure of a more quantitative nature than the essentially qualitative indices used previously.

Methods

The study combined data from two sources: (*1*) lung function information collected in 1980-1981 as part of a cross-sectional study investigating the evolution of lung function in the transition from adolescence to early adulthood in approximately 900 young adults without occupational exposure to dust or fumes (24) (hereafter referred to as the parent study), and (*2*) information on their lifetime environmental exposure to tobacco smoke and other home pollutants obtained by means of a questionnaire developed specifically for the purposes of the present study. The questionnaire was mailed during 1983 and 1984 to all participants of the parent study. Only subjects who reported never having smoked regularly before the date of the lung function tests were retained for analysis.

Study Population and Lung Function Data

The parent study has been reported in full elsewhere (24). In brief, subjects in the target age group (15 to 35 yr of age) were recruited on a volunteer basis from a school, a junior college, and two downtown Montreal banking institutions (table 1). They answered an interviewer-administered respiratory symptom questionnaire (ATS-DLD) (25) that included questions on smoking, and they performed the following lung function tests: (*1*) forced expiratory flow-volume curves with measurements of FVC, FEV_1, peak expiratory flow rate (PEFR), forced expiratory flow rate in the middle half of the FVC (FEF_{25-75}), and forced expiratory flow rates after 50 and 75% of FVC had been expelled ($\dot{V}max_{50}$ and $\dot{V}max_{75}$); (*2*) single-breath diffusing capacity for carbon monoxide (D_{LCO}) with correction for back pressure calculated from carboxyhemoglobin ($COHb\%_a$) measured by an oxygen rebreathing technique; (*3*) FRC measured by a constant pressure volume displacement plethysmograph. The slow VC was also recorded to allow calculation of residual volume (RV) and TLC. Futher details on techniques, calculations, procedure, and selection of measurements used to characterize each participant's lung function are given in the earlier report (24).

(*Received in original form September 22, 1987 and in revised form March 1, 1988*)

[1] From the Department of Epidemiology and Biostatistics, McGill University, Montreal, Quebec, Canada.

[2] Supported by a grant from the Medical Research Council of Canada.

[3] Correspondence and requests for reprints should be addressed to Pierre Ernst, M.D., Pulmonary Research Laboratory, Department of Epidemiology and Biostatistics, McGill University, 1110 Pine Avenue West, Montreal, Quebec, Canada H3A 1A3.

[4] Recipient of Fellowship from the National Health Research Development Program.

[5] Career Investigator of the Medical Research Council of Canada.

TABLE 1

SOURCE OF THE STUDY POPULATION

Institution	Target*	Contacted†	Responded (n)	Responded (%)‡
Bank 1	420	372	303	81.4
Bank 2	229	225	163	72.4
College	120	109	84	77.1
High school	128	112	86	76.8
Total	897	818	636	77.8

* Studied in a cross-sectional respiratory survey carried out in 1980/1981 [24].

† Through the institution or a home address recorded at the time of the previous survey.

‡ Percentage of those contacted.

TABLE 2

CHARACTERISTICS OF THE 293 NONSMOKING SUBJECTS* IN WHOM THE EFFECTS OF ENVIRONMENTAL EXPOSURE TO TOBACCO SMOKE WERE STUDIED

	Men (n = 133)			Women (n = 160)		
	Mean	SD	Range	Mean	SD	Range
Age, yr	24.8	6.7	14–36	22.6	6.2	13–35
Height, cm	178.7	6.5	159–190	162.6	5.8	145–178
Weight, kg	72.8	11.0	52–108	56.6	8.0	36–90
COHb, %†	1.20	1.24	0–4.88	1.81	1.46	0–8.12
Cumulative exposure to smoke in the home (persons × years)	21.2	17.0	0–64.0	22.8	17.4	0–75.0
Cumulative exposure to smoke at work (packs/day × years)	1.9	3.0	0–17.1	2.0	3.6	0–23.8

* Answered no to the following questions: Have you ever smoked cigarettes? (No means less than 20 packs of cigarettes or 12 oz of tobacco in a lifetime, or less than 1 cigarette a day for 1 yr.) Have you ever smoked a pipe regularly? Have you ever smoked cigars regularly? (Questions 25A, 26A, and 27A of the ATS-DLD questionnaire [25].)

† Five women had values greater than 5 g%; these high values may have been due to incorrect reporting (the subjects were smokers), technical error of measurement, or heavy environmental exposure.

Environmental Exposures at Home and at Work

The questionnaire used to assess environmental exposures was divided into two parts: home exposures and work exposures. A separate section dealt with home exposures to cooking and heating fuels. An introductory letter asking the subjects to participate in a study on indoor air quality and lung function accompanied the questionnaire. To assist the subject's recall of past exposures, separate subsections of the questionnaire made up of the same series of questions dealt with the following seven life reference periods: 5 yr of age and less (preschool), 6 to 11 yr of age (elementary school), 12 to 17 yr of age (high school), 18 to 22 yr of age (college for some), and 23 to 27, 28 to 32, and older than 32 yr of age. For each time period, the subject was asked how many persons lived in the same house and how many smoked; if there were smokers, the relationship to the subject was ascertained, and the average daily cigarette use was established based on the following code: light, less than 10 cigarettes; moderate, less than 20; heavy, 20 or more. In addition, use of cigars or pipes by household members was ascertained.

In the case of bank employees, the exposure to environmental tobacco smoke at work was assessed. Subjects were asked to report on the habitual "smoke conditions" that were present in each "area" in which they had ever been employed, both at the bank and earlier in their career. If tobacco smoke was seen and/or smelled occasionally, the exposure was considered light; if such was usually the case, the exposure was labeled moderate; and if that was always so, the work exposure was considered heavy. In the case of the students, it was assumed that they were not exposed during classes. The final two sections of the questionnaire dealt with the personal smoking habits of the subject and certain respiratory symptoms (copies of the questionnaire are available on request).

Indices of Environmental Exposure to Tobacco Smoke at Home

The questionnaire yielded two indices of cumulative exposure. The first was obtained from the product of the number of household members who smoked and the number of years living in the same household as the subject (persons × years). The second index was obtained by summing the product of the number of packs smoked per day by each smoker in the household and the years he/she lived in the same home as the subject (packs per day × persons × years). Packs per day were calculated from the code used by the subject in the questionnaire to describe the family members' smoking habits as follows: light, moderate, and heavy smoking habits were assigned values of 5, 15, and 25 cigarettes per day, respectively; if unknown, i.e., if a family member smoked but it was not known how many cigarettes, a value of 7 cigarettes per day was assigned, and the total was converted to packs per day assuming 1 pack equals 20 cigarettes.

Indices of Exposure to Tobacco Smoke at Work

Bank employees were asked to assess smoke conditions in the work area as light, moderate, heavy, or unable to quantify. These qualitative assessments were arbitrarily converted to number of cigarettes as follows: light = 5, moderate = 15, heavy = 25, unable to quantify = 7. The total was divided by 20 to yield packs and multiplied by duration to yield pack-years of exposure at work. For calculations of both home and work exposure indices, exposure that occurred after the date of the lung function test was excluded.

Indices of Exposure to Home Cooking Fuels

The length of time living in homes using natural gas or electricity as a cooking fuel was calculated for each subject, yielding two indices of exposure to cooking fuel expressed in years of exposure.

Analysis

The contribution of the indices of environmental exposure to the prediction of lung function test results was examined using multiple linear regression (SAS statistical package, GLM procedure) (26). Men and women were analyzed separately. Each regression model contained age, height, Quetelet index ($100 \times \text{weight}/\text{height}^2$), respiratory pressures, and cumulative exposure to cooking fuels at home. When examining the effect of exposure at home, cumulative exposure at work was included in the regression equation. When examining for the effect of exposure at work, cumulative exposure at home (persons × years) was included in the predictive model.

Results

Two hundred ninety-three subjects in the parent study (133 men, 160 women) were considered to be nonsmokers according to their answers to the questionnaire administered at the time of the lung function tests. Other descriptive characteristics are shown in table 2, and the definition of a nonsmoker is made explicit in the footnote to this table; table 3 provides the mean lung function results for these subjects.

The principal study results are presented in table 4. Exposure at home (expressed as persons × years), and at work (expressed as packs/day × years) was similar in men and women. However, inverse relationships between lung functions and environmental exposure were found more often in men than in women.

For instance, in men there were inverse relationships between cumulative exposure to environmental tobacco smoke in the home (persons × years) and flows at low lung volumes (FEF_{25-75}, $p < 0.01$; $\dot{V}max_{50}$, $p < 0.05$ and less strong for $\dot{V}max_{75}$, $p = 0.06$). Similar relationships were found when the cumulative exposure at home was expressed as packs/day × persons × years (data not shown). However, the greater variability in this latter measure of exposure with larger standard errors reduced the level of statistical significance. When parti-

TABLE 3

LUNG FUNCTIONS IN 293 NONSMOKING SUBJECTS

Lung Function	Men (n = 133)* Mean	Men (n = 133)* SD	Women (n = 160)* Mean	Women (n = 160)* SD
Derivatives of the flow-volume curves				
FVC, L	5.18	0.73	3.80	0.55
FEV_1, L	4.49	0.63	3.23	0.48
PEFR, L/s	10.6	2.72	7.2	2.48
FEF_{25-75}, L/s	5.29	1.44	4.19	1.07
$Vmax_{50}$, L/s	6.36	1.86	5.01	1.18
$Vmax_{75}$, L/s	3.26	1.12	2.65	0.85
Lung volumes				
VC, L	5.42	0.78	3.72	0.64
RV, L	2.04	0.73	1.68	0.62
TLC, L	7.27	1.13	5.29	0.89
Diffusing capacity for CO (single breath), ml/min/mm	34.3	6.1	23.3	4.5

* Not all subjects completed all tests satisfactorily; the lowest numbers were for lung volumes (124 men, 148 women). FVC and FEV_1 were completed by all

tioning the home exposure according to the family member who smoked, an inverse relationship between the FEF_{25-75} and maternal smoking habits was also demonstrated with a regression coefficient for FEF_{25-75} of 0.04 L/s per pack/day per year that the mother smoked ($p < 0.05$). The effect of environmental tobacco smoke exposure in the different periods of life was also examined. In men, but not in women, a statistically significant inverse association between exposure before 17 yr of age and FEF_{25-75} was found. When the analyses were restricted to the exposure during the 5 yr immediately preceding the lung function tests, no such association was observed. A small decrease in RV with increasing cumulative exposure at home (persons × years) was also found in men. The cumulative exposure to environmental tobacco smoke at work was much lower than that at home. There was, however, an inverse relationship between the slow VC and increasing exposure at work in men ($p < 0.05$).

In women, there was no significant relationship between any of the lung functions measured and cumulative exposure to environmental tobacco smoke at home (persons × years or packs/day × persons × years). However, cumulative exposure at work (packs/day × years) showed a statistically significant inverse relationship to D_{LCO}, but had no effect on spirometric parameters or lung volumes.

Discussion

The present results suggest that environmental exposure to tobacco smoke during the growth period of the lungs, especially early in life, permanently affects their mechanical properties in young men (reflected in changes in derivatives of the flow-volume curve), whereas exposure to second-hand smoke at work affects the diffusing characteristics of the lung in young women. These findings complement published data implicating home exposure, particularly to mothers' cigarette smoking. Thus, inverse relationships between environmental exposure to tobacco smoke and parameters derived from the FVC maneuver (27) have been described in both sexes, though the relative effects in males and females vary in different studies, and there are also inconsistencies between studies as to relative deficits in large or small airways function (23). Our findings demonstrating mostly small airways abnormality in men are consistent with those of Taussig and coworkers (28, 29), who have reported differences in the mechanical properties of the lung with greater susceptibility to small airways obstruction in boys than in girls. Male-female differences similar to our own results have also been reported in young active smokers (30).

The effect of environmental exposure to tobacco smoke at work on the diffusing capacity of young women has not to our knowledge been previously documented. This is due at least in part to the lack of studies examining the long-term consequences of this exposure in the workplace (31). In a similar age group, Enjeti and coworkers (30) found decreases in diffusing capacity in relation to active smoking more prominent in females than in males. These sex differences may reflect distinct pathophysiologic responses to environmental agents, which may in turn contribute to the sex differences in the incidence of chronic airflow obstruction and primary pulmonary hypertension.

Mild reductions in some lung volumes (RV) in relation to exposure at home and at work (VC, TLC) were found in men only. These reductions in lung volumes may represent a decrement in lung growth analogous to that reported in children for FEV_1 in relation to environmental exposure to tobacco smoke (7). However, caution in interpretation is needed since the multiple tests of significance (involving both exposure and response measure-

TABLE 4

REGRESSION COEFFICIENTS OF LUNG FUNCTIONS ON INDICES OF CUMULATIVE EXPOSURE TO ENVIRONMENTAL TOBACCO SMOKE*

Exposure Index (units)	Men: Home (persons × years)	Men: Work (packs/day × years)	Women: Home (persons × years)	Women: Work (packs/day × years)
Mean (maximum)	21.2 (84.0)	1.9 (17.1)	22.8 (75.0)	2.0 (23.8)
Flow-volume curves				
FVC	0.001	−0.038	0.022	−0.009
FEV_1	−0.002	−0.022	−0.001	−0.011
PEFR	−0.010	−0.018	0.003	−0.025
FEF_{25-75}	−0.020§	0.024	−0.003	−0.023
$Vmax_{50}$	−0.020‡	0.015	0.003	−0.014
$Vmax_{75}$	−0.012†	0.012	−0.001	−0.007
Lung volumes				
VC	0.004	−0.045‡	0.001	−0.006
RV	−0.008‡	−0.023	−0.000	0.006
TLC	−0.003	−0.089†	−0.001	0.002
Diffusing Capacity	0.020	−0.202	−0.007	−0.258‡

* All regression coefficients are adjusted for age, height, Quetelet index, respiratory pressures, and cumulative exposure to cooking fuels at home; coefficients for exposure at home are adjusted for cumulative exposure at work; coefficients for exposure at work are adjusted for cumulative exposure at home

† $p < 0.10$

‡ $p < 0.05$

§ $p < 0.01$

ments) are likely to have resulted in some associations achieving statistical significance by chance. On the other hand, some of the associations suggest effects that are by no means insignificant. For instance, the FEF_{25-75} of a young man 20 yr of age who had never smoked and always been at home would be 800 ml less if both his parents smoked than if they did not. Similarly, a young woman who had never smoked but who had worked in an office for 10 yr where smoke was always seen or smelled would have a D_{LCO} 3 units lower than if she had worked in a smoke-free office.

In most previous studies containing subjects in a similar age group, no relation between lung function and environmental tobacco exposure was found (16, 18, 19). Reasons for positive findings in this study may include our use of a cumulative and essentially quantitative estimate of exposure rather than a qualitative one (22, 23) with, in consequence, a lessening of the attenuation of dose-response relationships that inevitably accompanies misclassification (32). The use of a questionnaire to assess a subject's exposure to second-hand smoke has been validated, at least for recent exposure (33). However, the assessment of past exposure by questionnaire has limitations (34) that are likely to have caused an underestimation of the actual lung function deficit attributable to second-hand smoke.

References

1. World Health Organization. Health aspects related to indoor air quality: Report on a WHO working group. EURO Reports and Studies 21. Geneva: WHO, 1979.
2. Shephard RJ. The risks of passive smoking. London: Groom Helm, 1982; 1-191.
3. Rogers B. Global per capita consumption of manufactured cigarettes - 1985. Chron Dis Canada 1986; 7:54-5.
4. Tager IB, Weiss ST, Rosner B, Speizer FE. Effect of parental cigarette smoking on the pulmonary function of children. Am J Epidemiol 1979; 110:15-26.
5. Weiss ST, Tager IB, Speizer FE, Rosner B. Persistent wheeze. Its relation to respiratory illness, cigarette smoking, and level of pulmonary function in a population sample of children. Am Rev Respir Dis 1980; 122:697-707.
6. Vedal S, Schenker MB, Samet JM, Speizer FE. Risk factors for childhood respiratory disease: analysis of pulmonary function. Am Rev Respir Dis 1984; 130:186-92.
7. Tager IB, Weiss ST, Munoz A, Rosner B, Speizer FE. Longitudinal study of the effects of maternal smoking on pulmonary function in children. N Engl J Med 1983; 309:699-703.
8. Taskin DP, Clark VA, Simmons M, *et al*. The UCLA population studies of chronic obstructive respiratory disease: relationship between parental smoking and children's lung function. Am Rev Respir Dis 1984; 129:891-7.
9. Hasselblad U, Humble CG, Graham MG, Anderson HS. Indoor environmental determinants of lung function in children. Am Rev Respir Dis 1981; 123:479-85.
10. Burchfiel CM, Higgins MW, Keller JB, Howatt WF, Butler WJ, Higgins ITT. Passive smoking in childhood: respiratory conditions and pulmonary function in Tecumseh, Michigan. Am Rev Respir Dis 1986; 133:966-73.
11. Ware JH, Dockery DW, Spiro A III, Speizer FE, Ferris BG Jr. Passive smoking, gas cooking and respiratory health of children living in six cities. Am Rev Respir Dis 1984; 129:366-74.
12. Berkey CS, Ware JH, Dockery DW, Ferris BG Jr, Speizer FE. Indoor air pollution and pulmonary function growth in pre-adolescent children. Am J Epidemiol 1986; 123:250-60.
13. Schilling RSF, Letai AD, Hui SL, Beck GJ, Schoenberg JB, Bouhuys A. Lung function, respiratory disease and smoking in families. Am J Epidemiol 1977; 106:274-83.
14. Dodge R. The effects of indoor air pollution on Arizona children. Arch Environ Health 1982; 37:151-5.
15. Lebowitz MD, Armet DB, Knudson R. The effect of passive smoking on pulmonary function in children. Environ Int 1982; 8:371-3.
16. Kauffmann F, Tessier JF, Oriol P. Adult passive smoking in the home environment: a risk factor for chronic airflow limitation. Am J Epidemiol 1983; 117:269-80.
17. White JR, Froeb WF. Small airways dysfunction in nonsmokers chronically exposed to tobacco smoke. N Engl J Med 1980; 302:720-3.
18. Comstock GW, Meyer MB, Helsing KJ, Tockman MS. Respiratory effects of household exposures to tobacco smoke and gas cooking. Am Rev Respir Dis 1981; 124:143-8.
19. Kentner M, Triebig G, Weltle D. The influence of passive smoking on pulmonary function. A study of 1351 office workers. Prev Med 1984; 13:656-69.
20. Samet JM, Marbury MC, Spengler JD. Health effects and sources of indoor air pollution. Part 1. Am Rev Respir Dis 1987; 136:1486-1508.
21. Tager IB, Segal MR, Munoz A, Weiss ST, Speizer FE. The effect of maternal cigarette smoking on the pulmonary function of children and adolescents. Analyses of data from two populations. Am Rev Respir Dis 1987; 136:1366-70.
22. Weiss ST, Tager IB, Schenker M, Speizer FE. The health effects of involuntary smoking. Am Rev Respir Dis 1983; 128:933-42.
23. Tager IB. "Passive smoking" and respiratory health in children. Sophistry or cause for concern? Am Rev Respir Dis 1986; 133:959-61.
24. Leech JA, Ghezzo H, Stevens D, Becklake MR. Respiratory pressures and function in young adults. Am Rev Respir Dis 1983; 128:17-23.
25. Ferris BG Jr. Epidemiology standardization project. Am Rev Respir Dis 1978; 118(2 part 2):1-120.
26. SAS Institute. SAS user's guide, 1979 edition. Raleigh: SAS Institute Inc., 1979.
27. U.S. Department of Health and Human Services. The health consequences of smoking: chronic obstructive lung disease. A report of the Surgeon-General, Washington, D.C.:U.S. Department of Health and Human Services, Public Health Service, Office on Smoking and Health, 1984 (DHHS (PHS) publication no. 84-50205, 363-410).
28. Taussig LM, Cota K, Kaltenborn W. Different mechanical properties of the lung in boys and girls. Am Rev Respir Dis 1981; 123:640-3.
29. Tepper RS, Morgan WJ, Cota K, Wright A, Taussig LM. Physiologic growth and development of the lung during the first year of life. Am Rev Respir Dis 1986; 134:513-9.
30. Enjeti S, Hazelwood B, Permutt S, Menkes H, Terry P. Pulmonary function in young smokers: male-female differences. Am Rev Respir Dis 1978; 118:667-76.
31. Bake B. Does environmental tobacco smoke affect lung function? Eur J Respir Dis [Suppl] 1984; 133:85-7.
32. Armstrong BG, Oakes D. Effects of approximation in exposure assessments on estimates of exposure-response relationships. Scand J Work Environ Health 1982; 8(Suppl 1:20-3).
33. Pershagen G. Validity of questionnaire data on smoking and other exposures with special reference to environmental exposure to tobacco smoke. Eur J Respir Dis [Suppl] 1984; 133:76-80.
34. Wald NJ, Boreham J, Bailey A, Ritchie C, Haddon JE, Knight G. Urinary cotinine as a marker of breathing other people's tobacco smoke. Lancet 1984; 1:230-1.